'Incessant' tachycardias in Wolff-Parkinson-White syndrome[1]

I: Initiation without antecedent extrasystoles or PR lengthening, with reference to reciprocation after shortening of cycle length

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In 6 patients with the Wolff-Parkinson-White (WPW) syndrome, repetitive, almost continuous (incessant) reciprocating atrioventricular (AV) tachycardia has been shown to arise when the sinus cycle length was shortened to a critical point, at which unidirectional block occurred without the classical feature of PR prolongation. Though this phenomenon superficially resembles an aspect of chronic intranodal reciprocating tachycardia of children, basic differences can be identified. It was encountered more frequently in younger subjects; the only patient over 45 developed the arrhythmia as a complication of therapy. This incessant mechanism may explain some cases in which antiarrhythmic treatment does not control reciprocating tachycardia in the WPW syndrome, but such a mechanism can also occur spontaneously.

Reciprocating atrioventricular (AV) tachycardias, whether involving the AV node alone or in association with an extranodal bypass, depend on the existence of two separate functional (or anatomic) pathways which can constitute a circuit that permits reciprocation—the persistent transmission of an impulse anterogradely in the one direction and retrogradely in the other. Classically, such tachycardias are initiated by extrasystoles that induce anterograde block in one pathway, with PR prolongation, and occur in the form of intermittent episodes of varying duration, usually infrequently, but sometimes sufficiently often to be labelled repetitive (Parkinson and Papp, 1947). It is only recently that a specific 'incessant' form of reciprocating AV nodal tachycardia has been recognized and its features clarified (Coumel et al., 1967; Coumel, 1975). This arrhythmia tends to affect children rather than adults, classically shows inverted P' waves in leads II, III, and a VF (with RP'

longer than P'R) and is initiated not by PR prolongation but by a critical shortening of the PP interval in sinus rhythm. We have now recognized that this last mechanism, viz. critical shortening of the PP interval, can induce reciprocating tachycardia in the Wolff-Parkinson-White (WPW) syndrome, especially, though not invariably, in response to the administration of various antiarrhythmic agents, and this report describes 6 cases that illustrate its features. Electrophysiological studies were carried out by recognized techniques of intracardiac recording and programmed stimulation (Curry, 1975), the patients or their guardians having given consent after proper explanation; sedation was not used and all medications had been discontinued for at least 72 hours before investigation.

Case reports

Case 1

A 2-year-old child suffered from intractable paroxysmal supraventricular tachycardia that occurred in incessant or apparently self-limiting attacks (Fig. 1), either spontaneously or brought on

by febrile illnesses. During sinus rhythm, the pattern of WPW conduction (Fig. 1) was usually absent. It was not possible to initiate supraventricular tachycardia by premature atrial or ventricular stimulation. Isoprenaline was then ad-ministered intravenously, and the initiation of attacks of tachycardia without antecedent PR prolongation is shown in Fig. 1; in Fig. 2 (in which the WPW pattern is now seen) this is confirmed and the tachycardia is seen to restart after very subtle
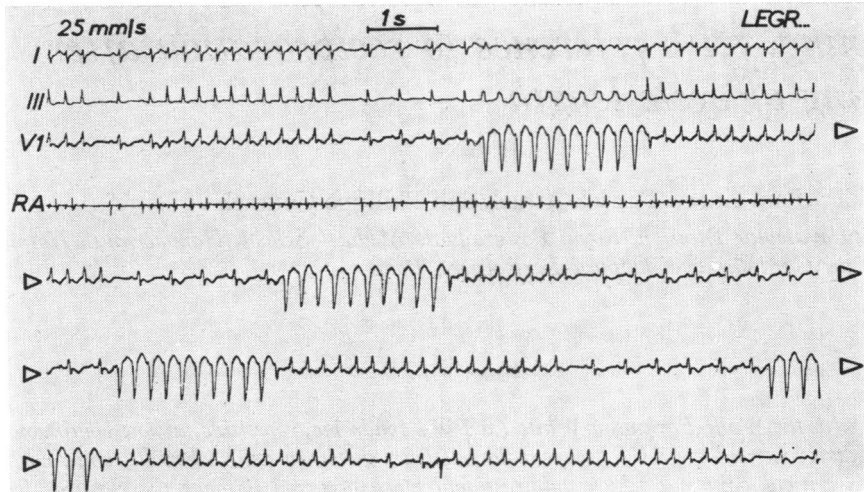

FIG. 1   *Case 1: recorded after the intravenous administration of isoprenaline shows three surface leads and a right atrial electrogram (RA). The third, fifth, sixth, and seventh tracings are continuous recordings of V1. Short paroxyms of tachycardia are seen to end spontaneously but to recur after two or more sinus beats; the PR intervals of the sinus beats immediately preceding the onset of tachycardia are not prolonged. In most instances, the first 7 to 10 beats of the tachycardia show QRS widening caused by left bundle-branch block which does not influence the rate of the tachycardia.*
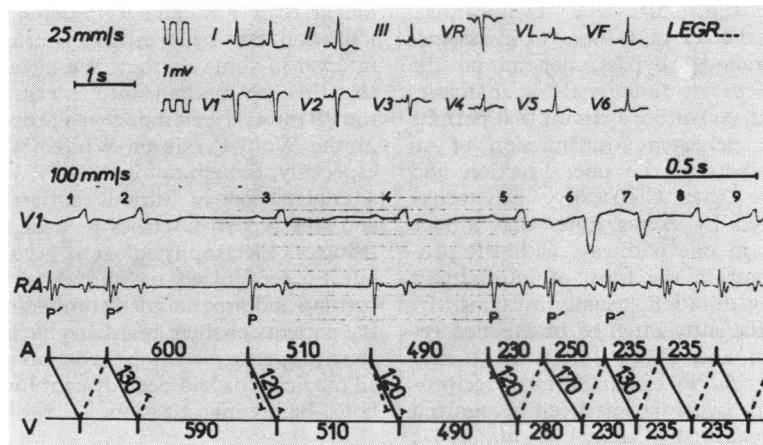

FIG. 2   *Case 1: the upper panel is a standard 12-lead electrocardiogram recorded one week before the study, showing the presence of the WPW syndrome type B (chest leads recorded at half voltage). The lower panel shows the same phenomenon as in Fig. 1, but displayed at faster paper speed: the PR interval in the third, fourth, and fifth beats is 120 ms, the P4-P5 interval being 20 ms shorter than the P3-P4 interval. The first RR interval in tachycardia (280 ms) was longer than the subsequent ones (235). In the right atrial electrocardiogram P and P′ are used synonymously with A for atrial activation.*

shortening of the cycle length in sinus rhythm. The first RR interval of tachycardia is relatively long (280 ms) and then becomes shorter. A likely explanation for failure of initiation of tachycardia by beats 3 and 4 (Fig. 2) is concealed anterograde conduction within the accessory pathway, but this is absent in beat 5 because of the shortening of the relevant PP interval.

Case 2

This patient was an 18-year-old boy with intermittent type A WPW syndrome associated with frequent attacks of reciprocating supraventricular tachycardia since the age of 1 year. While at first vagal manoeuvres often terminated the episodes, recently the attacks had occurred so frequently that half his waking day was spent in tachycardia.
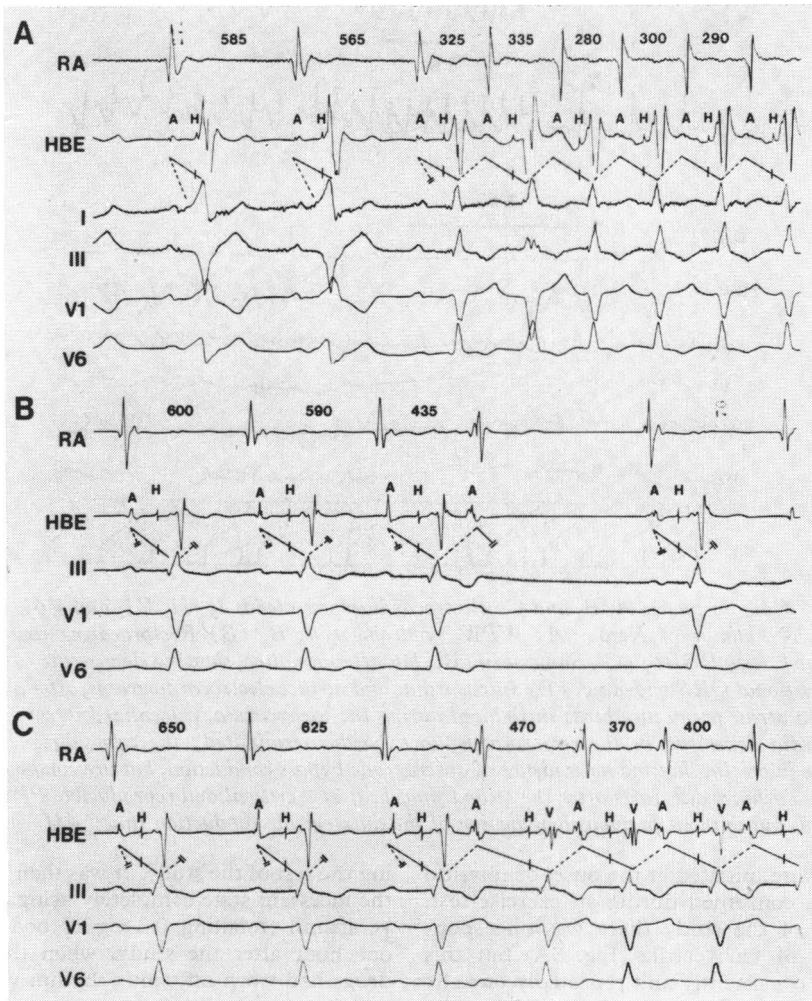

FIG. 3 Case 2: right atrial (RA) and His bundle (HBE) electrograms recorded simultaneously with leads I, III, V1, and V6. (A) There is a pre-excitation pattern in the first two complexes, with gradual shortening of the cycle length to 565 ms: tachycardia with narrow QRS complexes is initiated by the third complex. (B) Sinus rhythm, after intravenous ajmaline and verapamil: no evidence of pre-excitation; the third beat is followed by a spontaneous atrial echo which does not initiate tachycardia. (C) Soon after (B): the first three beats are sinus in origin, the third being followed by an atrial echo which now initiates tachycardia. Note that in both (A) and (C), the first RR interval in tachycardia was longer than those in subsequent cycles.
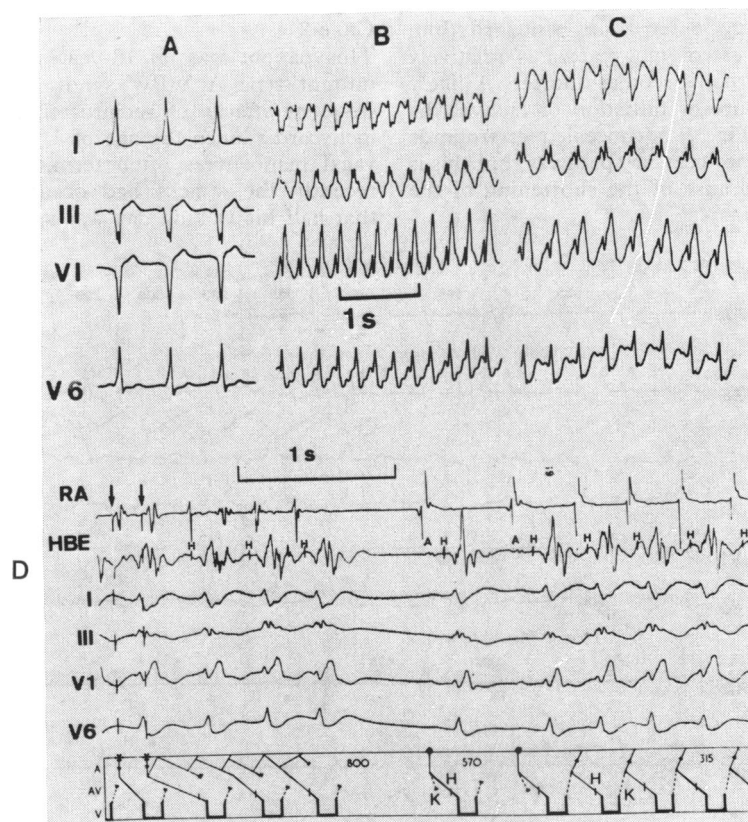
FIG. 4   *Case 3: panels A, B, and C: electrocardiograms (leads I, III, V1, and V6). H = His bundle; K = bundle of Kent. (A) WPW syndrome type B. (B) Reciprocating tachycardia (240/min), with RBBB. (C) Same as in (B), but after ajmaline, showing slower rate (180/min) with additional QRS widening. (D) Intracardiac and surface electrocardiograms, after ajmaline: after two atrial premature beats introduced during the tachycardia, a localized irregular atrial tachycardia developed as the reciprocating tachycardia terminated; the two subsequent beats are sinus in origin, showing no evidence of anterograde bypass conduction, but are conducted with RBBB. Tachycardia starts after the second sinus beat at a critical and reproducible PP interval of 570 ms but without previous lengthening of the anterograde conduction time (AH = 120 ms).*

Many attacks were initiated at the onset of physical activity, as was confirmed during an exercise test.

At the start of the study there was one spontaneous attack of tachycardia (Fig. 3A) but this occurred more consistently and repeatedly once he had received intravenous ajmaline (0·75 mg/kg body weight); when these were stopped with electric stimuli, only a few sinus beats followed before another episode of tachycardia ensued (Fig. 3C). After the addition of verapamil (0·15 mg/kg body weight) only one or two abortive atrial echo beats occurred (Fig. 3B). Once ajmaline had been given, there was anterograde bypass block, with no evidence of pre-excitation (Fig. 3B and C), and anterograde bypass conduction did not return dur-

ing the rest of the study. It was then possible to stop the incessant state completely using a larger dose of verapamil (totalling 0·3 mg/kg body weight), and one hour after the study, when the effect of the drugs had worn off, sinus rhythm was maintained.

Subsequently it has proved possible to suppress his tachycardia with verapamil, 400 mg, and long-acting quinidine, 2000 mg, a day, so that he now has attacks infrequently (once or twice a month) and for brief periods, despite active physical work.

Case 3

A 42-year-old man had suffered from increasingly frequent attacks of reciprocating supraventricular tachycardia for 3 years: electrocardiogram showed

the WPW syndrome type B (Fig. 4A). He invariably showed right bundle-branch block during the reciprocating tachycardia, and in sinus rhythm when the bypass was blocked by ajmaline (Fig. 4B, C, and D). Incremental atrial pacing up to a rate of 220/min failed to initiate tachycardia, but this was induced by single atrial extrastimuli in the range of 290 ms after the last preceding driven beat; the effective refractory period of the bypass was 290 ms and of the AV node, 270 ms.

He was then given ajmaline intravenously (0·75 mg/kg body weight). During the 20 minutes in which its effects persisted, it was impossible to suppress tachycardia for more than a few seconds; thereafter he lost the incessant tachycardia. As can be seen in Fig. 4D, the tachycardia was not preceded by any lengthening or anterograde conduction through the AV node, but started after shortening of the PP intervals to a critical point.

Subsequently he has remained free from tachycardia on verapamil 160 mg and long-acting quinidine 1000 mg a day.

Case 4

A 31-year-old man presented with recurrent reciprocating tachycardia of 4 years' duration. Electrocardiogram (Fig. 5A) showed the WPW syndrome type B. His typical tachycardia pattern is shown in Fig. 5B; in Fig. 5C the tachycardia had been slowed by ajmaline. During an electrophysiological study, incremental right atrial pacing showed a delta wave, and at a rate of 220/min, anterograde conduction in the bypass failed, reciprocation initiating orthograde tachycardia with narrow QRS com-

plexes; the refractory period of the bypass for anterograde conduction was 280 ms. After ajmaline (0·75 mg/kg body weight) intravenously, anterograde conduction down the bypass was blocked, and thereafter there were frequent spontaneous attacks of tachycardia by the incessant mechanism, without antecedent prolongation of anterograde conduction in the AV node (Fig. 6). During sinus rhythm, after ajmaline, the anomalous conduction was suppressed (Fig. 5D). When the effects of ajmaline were more pronounced (Fig. 6A and B), tachycardia was incessant and there was no anterograde bypass conduction; as these effects wore off, alternate beats were conducted anterogradely via the AV node and the bypass, and the tachycardia restarted less and less often, and finally could only be reinitiated by premature stimuli (Fig. 6C). He has responded well to prophylactic treatment with verapamil.

Case 5

A 5-year-old boy had suffered from recurrent paroxysmal tachycardia since the age of 3 months. Electrocardiograms taken in infancy showed no evidence of pre-excitation (Fig. 7B) but at the age of 4, the WPW syndrome type B became evident intermittently (Fig. 7A), and the tachycardia, which had been almost permanent (Fig. 8B) occurred less often but was disabling at times. The spontaneous cessation of tachycardia is shown in Fig. 8A, with no obvious anterograde PR prolongation or shortening of PP intervals prior to the reinitiation of tachycardia. The incessant nature of the tachycardia can be seen from Fig. 8B, only occasional sinus beats separating runs of arrhythmia. During tachycardia
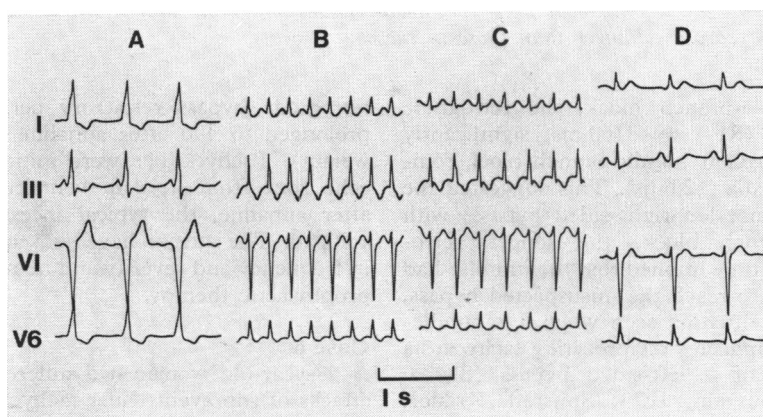
FIG. 5   *Case 4. Electrocardiograms leads I, III, V1, and V6. (A) Sinus rhythm with WPW syndrome type B (no medication). (B) Reciprocating tachycardia with narrow QRS complexes (215/min). (C) As in (B), rate slightly slowed by ajmaline (185/min). (D) A group of sinus beats after ajmaline, showing normalized conduction.*
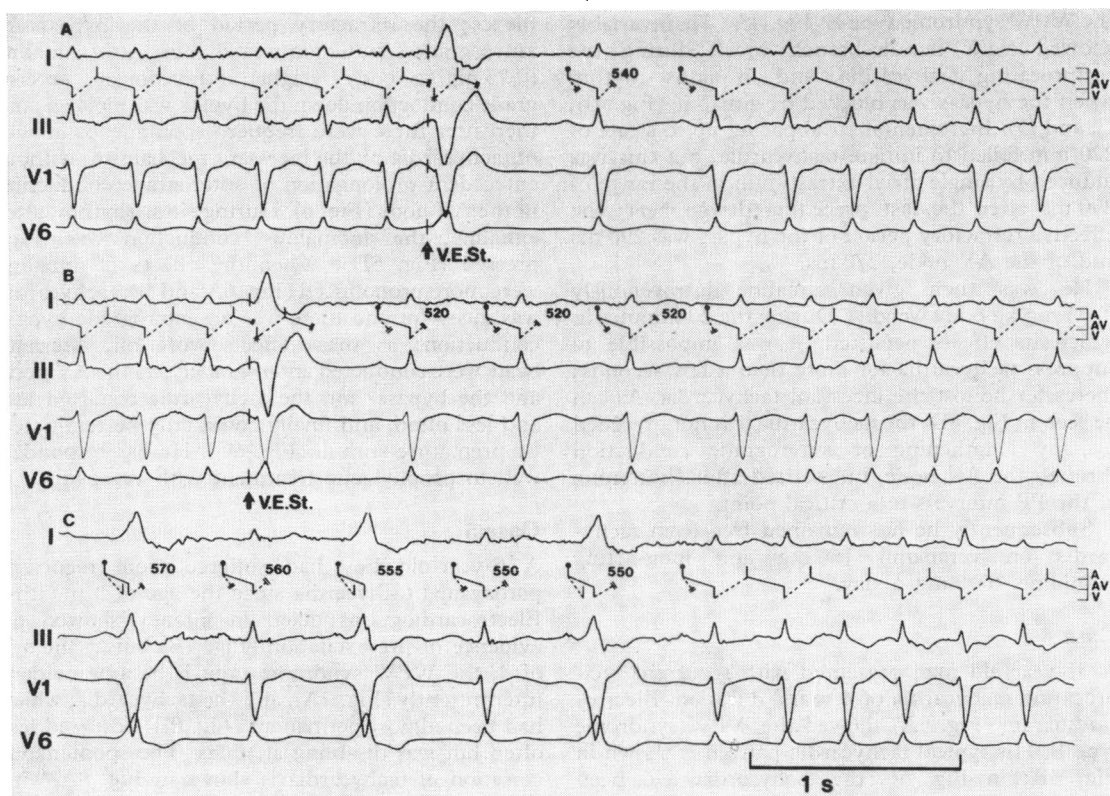
FIG. 6   *Case 4: Electrocardiograms (leads I, III, V1, and V6); (A) and (B) are within 10 minutes and (C) 15 minutes, after ajmaline. (A) Reciprocating tachycardia stopped by a ventricular extrastimulus; tachycardia reinitiated by the second of the subsequent sinus beats, neither of which showed pre-excitation. (B) As in (A), but tachycardia reinitiated by fourth sinus beat. (C) In sinus rhythm there is alternating nodal/bypass conduction, with reinitiation of tachycardia by a sinus beat with AV nodal conduction. Note that the first RR interval during tachycardia is longer than those in subsequent beats.*

with right bundle-branch block, the retrograde conduction time (RP') was 160 ms, significantly longer than during left bundle-branch block complicating tachycardia (120 ms). This suggested the presence of a concealed right-sided bypass: with right bundle-branch block, the longer retrograde conduction time implied that the impulse had to travel further, to reach the unsuspected bypass, analogous to the situation seen when left bundle-branch block complicates reciprocating tachycardia in the presence of a left-sided bypass (Slama, Coumel, and Bouvrain, 1973; Spurrell, Krikler, and Sowton, 1974).

At electrophysiological study the presence of intermittent WPW syndrome type B was noted, but the bypass refractory period was long (over 600 ms) and it ceased to conduct anterogradely. The

retrograde bypass refractory period was 280 ms, prolonged to 490 after ajmaline (0·7 mg/kg body weight). Tachycardias were initiated over a wide echo zone from atrial or ventricular extrastimuli; after ajmaline, the typical incessant pattern developed. The attacks have subsequently decreased in frequency and severity and he no longer requires prophylactic therapy.

Case 6
A 58-year-old woman had suffered from disabling attacks of supraventricular tachycardia for 4 years. Electrocardiograms in sinus rhythm revealed no definite evidence of pre-excitation, but the PR interval was 0·12 s. She was referred because an attack of tachycardia had not responded to combined therapy with digoxin, lignocaine, and
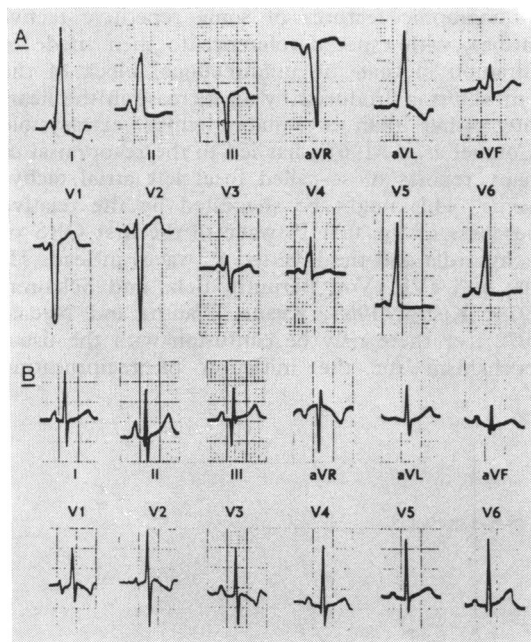
FIG. 7 Case 5. Twelve-lead electrocardiograms. (A) WPW syndrome type B, taken at the age of 4. (B) Tracing taken in infancy, showing no pre-excitation.

practolol (Fig. 9). As can be seen in Fig. 9b and c, the tachycardia stopped, but restarted as the sinus cycle length shortened. Electrophysiological study showed her to have a short AH interval as well as a left-sided bypass (bundle of Kent) as shown by the fact that, during induced tachycardia, the left atrium was depolarized 75 ms before the right (Fig. 10). The AH interval did not lengthen significantly on atrial pacing or with atrial extra-stimuli, and thus had features compatible with a partial atrionodal bypass ('James' fibres), which may explain the rapidity of the tachycardia (Fig. 9a). As the tachycardia reverted to sinus rhythm after intravenous verapamil, the cycle lengths alternated (Spurrell, Krikler, and Sowton, 1973; Curry and Krikler, 1976). She has had no attacks while on verapamil 360 mg daily.

Discussion

Classically, in junctional reciprocating tachycardia (AV nodal tachycardia), whether involving the AV node alone or as a feature of the WPW syndrome, the attacks are initiated by extrasystoles. The underlying prerequisite for the occurrence of a circus movement tachycardia is the presence of an avail-

able circuit in which the impulse can travel between atria and ventricles, and when the AV node alone is involved, the pathways are conventionally called α and β, reflecting differences in conduction velocity and refractory periods (Mendez and Moe, 1966; Janse et al., 1971). For tachycardia to start, unidirectional block is required in the β pathway, and conduction must be slowed so that the impulse traversing the α pathway can return retrogradely via β and then encounter a no-longer-refractory α pathway so that the re-entry process can continue. Initiation of tachycardia has been shown to follow premature beats induced in the atrium or ventricle that enter the circuit and cause unidirectional block in the α pathway and slowed conduction; this is seen as prolongation of the PR interval at the start of the tachycardia (Coumel et al., 1967, 1970; Goldreyer and Damato, 1971; Goldreyer, Weiss, and Damato, 1971; Wellens, 1971). The analogous mechanism has of course been shown to underly reciprocating tachycardias in the WPW syndrome; appropriately-timed atrial extrasystoles that are blocked in the anomalous pathway but conducted down the normal route characteristically initiate a circus movement
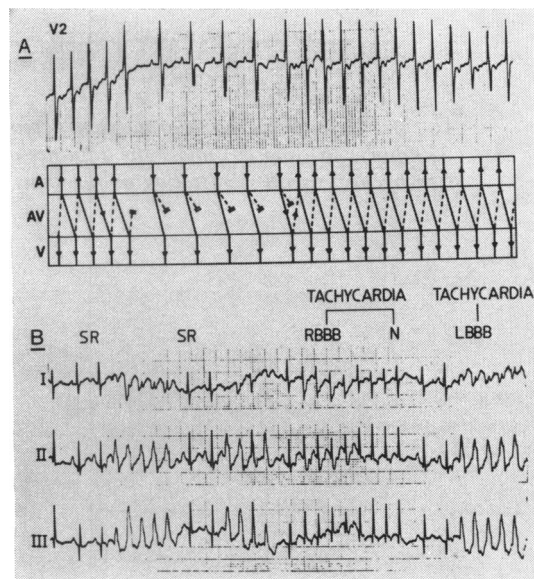

FIG. 8 Case 5. (A) Cessation and resumption of tachycardia with normal intraventricular conduction. (B) Incessant tachycardia, with repetitive changes from sinus rhythm (SR) to tachycardia which showed normal intraventricular conduction (N) and right and left bundle-branch aberration (RBBB and LBBB). During RBBB the RP' interval was 160 ms, and during LBBB, 120 ms.

tachycardia (Durrer *et al.*, 1967). Assessment of tachycardias initiated in this way is a fundamental investigation in the electrophysiological assessment of junctional reciprocating tachycardias (Wellens, 1971; Curry, 1975). The occurrence of appropriately-timed spontaneous extrasystoles is clearly an important factor in the occurrence of many cases of repetitive paroxysmal junctional tachycardias (Gallavardin, 1922; Parkinson and Papp, 1947; Krikler, 1974a).

Analysing conventional electrocardiograms, Katz and Pick (1956) and Kistin (1965) attributed repetitive paroxysmal tachycardia to a re-entry mechanism. Recognition of the characteristic electro-

cardiographic features of some repetitive tachycardias, with especial reference to their mode of initiation in that the unidirectional block of the pathway was produced by an increase in the heart rate rather than a suitably timed extrasystole (Coumel *et al.*, 1967), has led to the reappraisal of many reports of so-called focal left atrial tachycardia. This might be suggested by the relative closeness of the first P' wave to the first QRS of tachycardia and the inverted P' waves in leads II, III, and aVF (Von Bernuth, Belz, and Schairer, 1973; Kistin, 1965; Keane, Plauth, and Nadas, 1972); or there may be confusion with the usual mechanism for the initiation of reciprocating
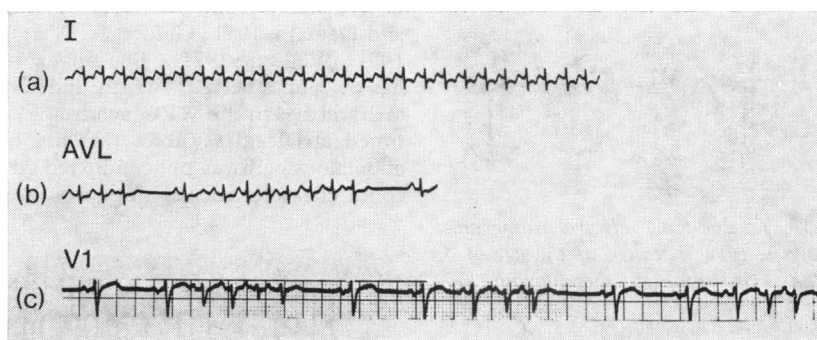

FIG. 9 *Case 6: Electrocardiograms recorded during tachycardia (a) before treatment (250/min). (b) and (c) under the influence of digoxin, practolol, and lignocaine: note cessation of tachycardia with reinitiation as the sinus cycle shortened, best seen in (c).*
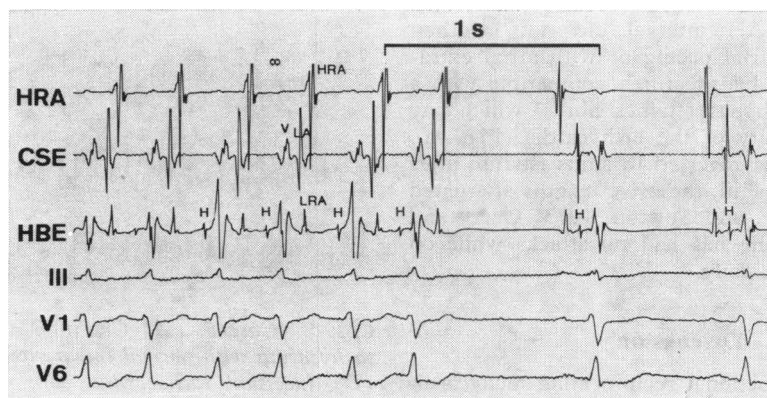

FIG. 10 *Case 6: Intracardiac and surface electrocardiograms, showing termination of tachycardia after intravenous verapamil. In tachycardia, the left atrium (LA in CSE) was depolarized 75 ms before the low right atrium (LRA in HBE); the normal atrial activation sequence is seen in the two subsequent sinus beats. AH=55 ms. CSE=coronary sinus electrogram.*

junctional tachycardia (Gettes and Yoshonis, 1970; Rosen, 1973). Using progressively accelerated atrial pacing, it is possible to show that a critical PP interval can be reached in order to initiate the tachycardia (Coumel et al., 1974; Coumel, 1975). Castellanos and Myerburg (1975) may well be hinting at this mechanism when they indicate that conspicuous sinus arrhythmia may act like extrasystoles in inducing reciprocating AV tachycardia.

Our cases with pre-excitation differ in several important ways from these chronic intranodal tachycardias (Coumel, 1975). Most important, the P'R interval was always longer than RP', instead of the converse, and RP' was constant and uninfluenced by vagal or drug effects. The relatively short and constant RP' in our present cases conforms with the use of a bypass tract retrogradely during tachycardia. Also, the first cycle length (RR) of tachycardia in Cases 1, 2, and 4 was longer than the cycle lengths in successive beats, by which time anterograde intranodal conduction had speeded in proportion to the heart rate; the first cycle length in the intranodal form is often shorter, in keeping

with the presence of an initial common pathway. Classically, of course, the intranodal tachycardias originate in infancy and persist for long periods, even decades; we now, however, describe briefer, perhaps ephemeral, phenomena, usually drug-induced.

In assessing mechanisms of the initiation of incessant tachycardia in the WPW syndrome, the same basic prerequisite obtains as with reciprocating tachycardias of the more usual variety affecting the AV junction or complicating the WPW syndrome: unidirectional block must exist in one pathway. Under the present circumstances, this can occur in the two possible ways illustrated in Fig. 11. If the refractory period of the accessory pathway is long in the anterograde direction, all that may be required for the sinus impulse to meet this situation may be a minor increase in the heart rate (shortening of sinus cycle length) so that the sinus impulse is conducted to the ventricles slowly via the AV node, as can be seen in the fifth sinus beat in Fig. 11A. Alternatively, anterograde unidirectional block in the accessory pathway may already exist,
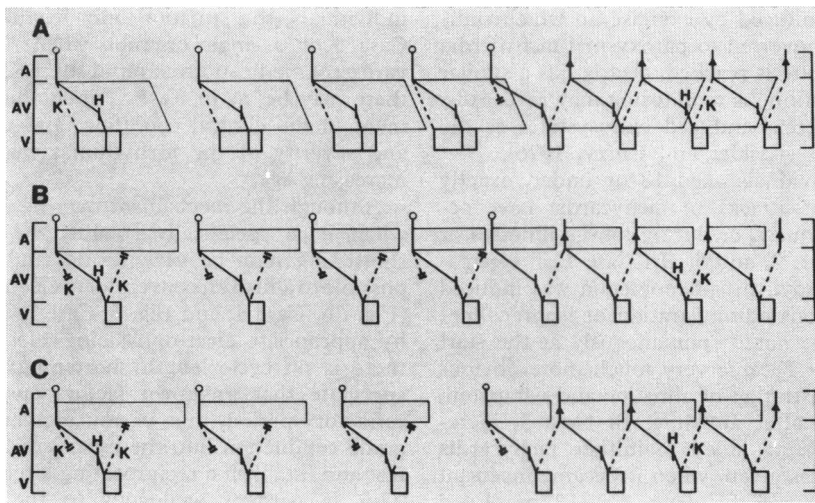

FIG. 11 Diagrammatic representation of conduction sequences in the initiation of incessant tachycardia. A=atrium; AV=AV junction, V=ventricle; K=anomalous pathway; H= bundle of His. Impulses commencing with open circles originate in the SA node. (A) initiation of tachycardia when shortening of sinus cycle length causes an impulse traversing the anomalous pathway after the fifth beat to fall in the refractory period of the preceding sinus beat; (B) consistent anterograde block in the bypass related to the refractory period of the atrium; when the sinus cycle length and atrial refractory period shorten, retrograde bypass conduction can initiate tachycardia, as occurs with the fifth sinus beat. (C) stable sinus rate: shortening of atrial refractory period is sufficient to enable previously blocked retrograde bypass conduction to penetrate fully after the fourth sinus beat and initiate tachycardia.

the pre-excitation being latent or concealed in the anterograde direction (Krikler, 1975) as is seen in the sinus beats in Fig. 11B. The second prerequisite follows from this: the time for conduction of the sinus beat through the AV node, when there is unidirectional block in the anomalous pathway, must be sufficiently long to permit the atrium to recover from activation by the preceding sinus beat; a possible corollary may be the need for the accessory pathway to recover from concealed anterograde conduction from the preceding sinus beat, if this had occurred.

Should these conditions be met, an atrial echo may occur when the sinus rate speeds up sufficiently for the refractory period of the anomalous pathway to be reached in the anterograde direction, this pathway now being conducted retrogradely (Fig. 11A); this may allow a burst of tachycardia to occur whenever the sinus cycle length is sufficiently short. On the other hand, when there is established unidirectional block in the anterograde pathway, speeding of the sinus rate may shorten the atrial and anomalous pathway refractory periods sufficiently to permit retrograde conduction up the anomalous pathway to the atrium, with an echo beat and tachycardia (Fig. 11B). The reciprocating AV tachycardia (whether intranodal or complicating pre-excitation) induced by exercise, in which sinus tachycardia is converted to paroxysmal tachycardia when a critical rate is reached, clearly has a similar mechanism; continuous monitoring may reveal this and exclude exercise-induced extrasystoles as the initiating factors (Krikler and Curry, 1976).

In the 3 individuals aged 18 or under, exactly similar incessant attacks of tachycardia have occurred spontaneously, or during febrile illnesses or exertion (Cases 1, 2, and 5). In Case 1, at electrophysiological study, this phenomenon was induced by the intravenous administration of isoprenaline; in Case 2 it was noted spontaneously at the start of the study but became very much more obvious after the administration of ajmaline and verapamil, and in Case 5, after ajmaline. In Case 3, incremental atrial pacing failed to initiate tachycardia until ajmaline was given, when it became incessant during the effect of the drug. Case 4 presented similar features in that, under the influence of ajmaline, which abolished anterograde bypass conduction, tachycardia was incessant; after its effect had worn off, premature stimuli were needed in order to initiate tachycardia. The classical features of incessant tachycardia were seen in Case 6 when she received vigorous antiarrhythmic therapy; the situation was not duplicated with ajmaline and verapamil during electrophysiological study.

For the electrocardiographic diagnosis of the WPW syndrome to be made in sinus rhythm, it is necessary for both the normal and anomalous pathways to function anterogradely, thus producing the characteristic fusion beat of the WPW syndrome. It is now becoming more and more obvious that in many cases there is intermittent or even permanent anterograde block in the anomalous pathway, causing the syndrome to be intermittent or indeed latent or concealed (Wilson, 1915; Slama et al., 1973; Spurrell et al., 1974). Unless this is appreciated, it is possible for cases of incessant junctional tachycardia to be considered to be AV nodal in origin: only Cases 3 and 4 consistently showed evidence of the WPW syndrome during sinus rhythm, it being intermittent in Cases 1, 2, and 5 (only appearing at the age of 4 in the latter even though tachycardia was present from early infancy). Case 6 never showed evidence of anterograde bypass conduction on surface electrocardiograms, electrophysiological investigation being necessary; we feel that the demonstration of an alternating cycle length during termination of the tachycardia had pointed to the presence of a bypass (Spurrell et al., 1973; Curry and Krikler, 1976). As is the case in incessant AV nodal reciprocating tachycardias, cycle-length-dependent reciprocation in the WPW syndrome mainly affects younger individuals, the situation only having occurred in Case 6 on a single occasion when she received a variety of medications in rapid succession. However, there may be, as in Case 5, spontaneous improvement in the clinical condition, and the frequency and severity of the arrhythmias may wane with increasing age.

Although the mechanism may be easy to detect when it is specifically sought, the cycle length shortening may be very subtle, and perhaps impossible to affirm on conventional electrocardiograms (Fig. 6b, Case 4, and Fig. 8, Case 5) but be shown by appropriate electrophysiological studies. When there is no cycle length shortening one can only speculate that unknown factors curtail the atrial refractory period, thus permitting concealed retrograde conduction into the bypass to become manifest and establish a reciprocating circuit (Fig. 11C). This is perhaps analogous to the finding that atropine permitted re-entry to become established at an AH interval that had not provoked tachycardia before it had been given (Akhtar et al., 1975); they diagnosed intranodal reciprocation but perusal of their Fig. 1, 2, and 3 suggests that their first two cases may have concealed pre-excitation.

As can be seen from our cases, the incessant mechanism may be brought to light or aggravated by antiarrhythmic agents. We have studied 5 of our patients using verapamil as an agent with pre-

dominant action on the AV node (Krikler, 1974b) and ajmaline as typical of agents that preponderantly depress myocardial (and thus bypass) conduction (Puech et al., 1964). Clearly we were able to influence the respective refractory periods in such a way that the incessant situation could be achieved, on some occasions with ajmaline alone, and on others when they were given in combination. In Case 6 this effect was not achieved during study, but the incessant situation was produced by antiarrhythmic polypharmacy. We feel that in some cases of reciprocating tachycardia complicating the WPW syndrome, apparent failure to respond to therapy may reflect the fact that the agents enhance the discrepancy between the respective refractory periods of the AV node and bypass and thus enhance the opportunity for tachycardia to occur. This should be borne in mind and the patient assessed by thorough electrophysiological study before it is concluded that antiarrhythmic therapy with drugs has failed, and the patient be subjected to surgical treatment or have a pacemaker implanted. In one of our patients (Case 2) large doses of both verapamil and quinidine were required before the arrhythmias could be effectively suppressed, and we believe that remote monitoring is useful in assessing the response of such cases and enabling the pattern of initiation of arrhythmia to be seen (Coumel et al., 1975).

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